

L Number	Hits	Search Text	DB	Time stamp
1	132	transgene and (e1a with e1b)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/11/16 17:43
2	127	transgene and (e1a with e1b) and promoter	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/11/16 17:43
3	316	(e1a with e1b) and promoter	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/11/16 17:43
4	127	(e1a with e1b) and promoter and transgene	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/11/16 17:43
5	520	e1a and e1b	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/11/16 17:44
6	95	(e1a and e1b) with vector	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/11/16 17:44
7	37	((e1a with e1b) and promoter and transgene) and ((e1a and e1b) with vector)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/11/16 17:44
-	0	(AAV and adenovir?) with hybrid	USPAT; IBM TDB	2001/01/31 09:34
-	113	(host adj cell?) and rep and cap	USPAT; IBM TDB	2001/01/31 09:36
-	0	((host adj cell?) and rep and cap) and adeno?	USPAT; IBM TDB	2001/01/31 09:36
-	7	((host adj cell?) and rep and cap) and ((induc? or comstitutiv?) with promoter?)	USPAT; IBM TDB	2001/01/31 09:41
-	15	((host adj cell?) and rep and cap) and E1a and E1b and E2a	USPAT; IBM TDB	2001/01/31 09:54
-	0	((host adj cell?) and rep and cap) and E1a and E1b and E2a	EPO; JPO; DERWENT	2001/01/31 09:54
-	254	helper with virus	EPO; JPO; DERWENT	2001/01/31 09:55
-	1268	helper with virus	USPAT; IBM TDB	2001/01/31 09:56
-	0	((((host adj cell?) and rep and cap) and E1a and E1b and E2a) not (helper with virus)	USPAT; IBM TDB	2001/01/31 09:56
-	741	helper adj virus	USPAT; IBM TDB	2001/01/31 09:56
-	0	((((host adj cell?) and rep and cap) and E1a and E1b and E2a) not (helper adj virus)	USPAT; IBM TDB	2001/01/31 09:57
-	3	((((host adj cell?) and rep and cap) and E1a and E1b and E2a) and transgene?	USPAT; IBM TDB	2001/01/31 09:58
-	0	((((host adj cell?) and rep and cap) and E1a and E1b and E2a) and transgene?	EPO; JPO; DERWENT	2001/01/31 09:58
-	15	((((host adj cell?) and rep and cap) and E1a and E1b and E2a) and (helper with virus)	USPAT; IBM TDB	2001/01/31 09:59

-	3	9534670.pn.	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/11/16 14:01
-	3	9615777.pn.	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/11/16 14:01
-	1	6258595.pn.	USPAT; IBM	2001/11/16 17:14
-	70	E1a adj promoter	TDB USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/11/16 17:14
-	20	E2a adj promoter	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/11/16 17:14
-	27	E1b adj promoter	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/11/16 17:15
-	1	(E1a adj promoter) and (E2a adj promoter) and (E1b adj promoter)	USPAT; US-PGPUB; EPO; JPO; DERWENT; IBM TDB	2001/11/16 17:22

FILE 'MEDLINE' ENTERED AT 16:59:07 ON 16 NOV 2001

L1 49 S E1A(W) PROMOTER
L2 43 S E2A(W) PROMOTER
L3 1 S E2B(W) PROMOTER
L4 34 S E1B WITH PROMOTER
L5 0 S L4 AND L1
L6 29 S E1A AND E1B AND E2A
L7 84905 S PROMOTER?
L8 16156 S L7 AND 6
L9 9 S L7 AND L6
L10 27 S E1B(W) PROMOTER
L11 0 S L1 AND L10 AND L2
L12 0 S L1 AND L10
L13 0 S L1 AND L2
L14 0 S L10 AND L2
L15 1 S HYBRID AAV AND ADENOVIRUS
L16 0 S TRANSGENE AND REP AND CAP AND E1A AND E2A
L17 131 S TRANSGENE AND AAV
L18 61 S REP AND CAP
L19 7 S L17 AND L18
L20 19 S TRANSGENE AND ITR
L21 1 S L19 AND L20
L22 29 S E1A AND E1B AND E2A
L23 1273466 S INDUC?
L24 84905 S PROMOTER?
L25 1 S L22 AND L23 AND L24
L26 17 S E1A AND CMV
L27 3 S E2A AND CMV
L28 0 S L26 AND L27
L29 3 S HYBRID ADENOVIRUS AND AAV
L30 30 S E2A AND TRANSGENE
L31 453 S E1A AND E1B
L32 0 S L30 AND L31
L33 0 S L30 AND ITR
E GAO/AU
E GAO G/AU
L34 75 S E3
E GAO GUANGPING/AU
E GAO GUANGPING/AU
L35 2 S HYBRID AND L34

L18 ANSWER 7 OF 11 MEDLINE
 ACCESSION NUMBER: 95134094 MEDLINE
 DOCUMENT NUMBER: 95134094 PubMed ID: 7832644
 TITLE: Unusual splice sites in the **E1A-E1B**
 cotranscripts synthesized in adenovirus type 40-infected
 A549 cells.
 AUTHOR: Ishida S; Fujinaga Y; Fujinaga K; Sakamoto N; Hashimoto S
 CORPORATE SOURCE: Cancer Research Institute, Sapporo Medical College,
 Japan.
 SOURCE: ARCHIVES OF VIROLOGY, (1994) 139 (3-4) 389-402.
 Journal code: 8L7; 7506870. ISSN: 0304-8608.
 PUB. COUNTRY: Austria
 Journal; Article; (JOURNAL ARTICLE)
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 199502
 ENTRY DATE: Entered STN: 19950307
 Last Updated on STN: 19970203
 Entered Medline: 19950222

AB The adenovirus E1 DNA region consists of two transcription units, E1A and
 E1B. In this paper we report that the **E1A-E1B**
 cotranscripts containing sequences of both the E1A and E1B regions are
 synthesized during adenovirus type 40 (Ad40) infection of A549 cells.
 Cytoplasmic RNA was isolated from Ad40-infected A549 cells at 24, 72, and
 100 h post infection (p.i.). The complementary (c) DNA was synthesized by
 reverse transcription using an oligo-dT primer and then amplified by the
 polymerase chain reaction (PCR) using primers derived from the E1A and
 E1B
 regions. The cDNAs thus amplified were sequenced either directly or after
 cloning into bacteriophage M13 **vectors**. Analysis of cDNA
 indicated that the **E1A-E1B** cotranscripts are
 synthesized at 72 h p.i., but not at 24 or 100 h p.i. Nucleotide
 sequences
 of three cDNAs of the **E1A-E1B** cotranscripts indicated
 that the cotranscripts originate from the E1A promoter and lack sequences
 for both the E1A poly(A) site and E1B cap site. The splices create open
 reading frames for **E1A-E1B** fused polypeptides around
 the **E1A-E1B** junctions in these mRNAs. Most
 interestingly, the sequence analysis showed that the 5' and 3' splice
 junctions in the two **E1A-E1B** cotranscripts do not
 conform to the splice consensus GT-AG rule. Our results thus suggest that
 factor(s) which lead to unusual splicing in the E1 mRNAs are present in
 Ad40-infected A549 cells.

L18 ANSWER 3 OF 11 MEDLINE
 ACCESSION NUMBER: 1998105729 MEDLINE
 DOCUMENT NUMBER: 98105729 PubMed ID: 9444984
 TITLE: Production and characterization of improved adenovirus
vectors with the E1, E2b, and E3 genes deleted.
 AUTHOR: Amalfitano A; Hauser M A; Hu H; Serra D; Begy C R;
 Chamberlain J S
 CORPORATE SOURCE: Department of Pediatrics, Duke University Medical Center,
 Durham, North Carolina 27710, USA.. amalf001@mc.duke.edu
 SOURCE: JOURNAL OF VIROLOGY, (1998 Feb) 72 (2) 926-33.
 Journal code: KCV; 0113724. ISSN: 0022-538X.
 PUB. COUNTRY: United States
 LANGUAGE: English
 FILE SEGMENT: Priority Journals
 ENTRY MONTH: 199802
 ENTRY DATE: Entered STN: 19980226
 Last Updated on STN: 19980226
 Entered Medline: 19980218

AB Adenovirus (Ad)-based **vectors** have great potential for use in
 the gene therapy of multiple diseases, both genetic and nongenetic. While
 capable of transducing both dividing and quiescent cells efficiently, Ad
vectors have been limited by a number of problems. Most Ad
vectors are engineered such that a transgene replaces the Ad
E1a, E1b, and E3 genes; subsequently the
 replication-defective **vector** can be propagated only in human 293
 cells that supply the deleted E1 gene functions in trans. Unfortunately,
 the use of high titers of E1-deleted **vectors** has been repeatedly
 demonstrated to result in low-level expression of viral genes still
 resident in the **vector**. In addition, the generation of
 replication-competent Ad (RCA) by recombination events with the E1
 sequences residing in 293 cells further limits the usefulness of
 E1-deleted Ad **vectors**. We addressed these problems by isolating
 new Ad **vectors** deleted for the E1, E3, and the E2b gene
 functions. The new **vectors** can be readily grown to high titers
 and have several improvements, including an increased carrying capacity
 and a theoretically decreased risk for generating RCA. We have also
 demonstrated that the further block to Ad **vector** replication
 afforded by the deletion of both the E1 and E2b genes significantly
 diminished Ad late gene expression in comparison to a conventional
 E1-deleted **vector**, without destabilization of the modified
vector genome. The results suggested that these modified
vectors may be very useful both for in vitro and in vivo gene
 therapy applications.

ACCESSION NUMBER: 83307850 MEDLINE
DOCUMENT NUMBER: 83307850 PubMed ID: 6615164
TITLE: Adenoviruses and human tumors: regulation of eukaryotic
chromatin structure?.
AUTHOR: Berencsi G; Nasz I
SOURCE: ARCHIV FUR GESCHWULSTFORSCHUNG, (1983) 53 (3) 239-52.
Journal code: 746; 0372411. ISSN: 0003-911X.
PUB. COUNTRY: GERMANY, EAST: German Democratic Republic
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198310
ENTRY DATE: Entered STN: 19900319
Last Updated on STN: 19900319
Entered Medline: 19831021

AB Adenoviruses possess four early gene clusters. **E1A**, and
E1B sequences are directly involved in the initiation of
transformation of both rodent, and human cells. Oncogenic potential of
the
in vitro immortalized rodent cells is also dependent on the expression of
E1A, and **E1B**. Genes **E2A**, and **E2B** are
coding for DNA-binding proteins. These regions may influence the
frequency
of in vitro transformation if complete virions were used for the
immortalization of rodent cells. The existence of the E3 gene cluster,
which has been shown to be non-essential to the productive replication in
certain host cells, is unique for the adenovirus family. Foreign DNA
fragments inserted into E3 or replacing it may modify the host range of
adenovirus, and may initiate oncogenic transformation of rodent cells.
May
cellular oncogenes replace the non-essential region of the adenoviral
genome? Are host-range modifications, the formation of defective genomes,
and interactions of viral, and cellular DNA in vivo biological properties
of adenoviruses? These questions are discussed in the light of recent
findings concerning virus-coded functions which may modify chromatin
structure, and may be associated with oncogenic potential of adenoviruses
in the natural hosts.

L21 ANSWER 12 OF 33 MEDLINE

ACCESSION NUMBER: 1998285701 MEDLINE

DOCUMENT NUMBER: 98285701 PubMed ID: 9621003

TITLE: Characterization of **wild-type** adeno-associated virus type 2-like particles generated during **recombinant** viral vector production and strategies for their elimination.

AUTHOR: Wang X S; Khuntirat B; Qing K; Ponnazhagan S; Kube D M; Zhou S; Dwarki V J; Srivastava A

CORPORATE SOURCE: Department of Microbiology and Immunology, Indiana University School of Medicine, Indianapolis, Indiana 46202,

USA.

CONTRACT NUMBER: HL-48342 (NHLBI)

HL-53586 (NHLBI)

HL-58881 (NHLBI)

+

SOURCE: JOURNAL OF VIROLOGY, (1998 Jul) 72 (7) 5472-80.

Journal code: KCV; 0113724. ISSN: 0022-538X.

PUB. COUNTRY: United States

Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199807

ENTRY DATE: Entered STN: 19980713

Last Updated on STN: 19980713

Entered Medline: 19980701

AB The pSub201-pAAV/Ad plasmid cotransfection system was developed to eliminate homologous **recombination** which leads to generation of the **wild-type** (wt) adeno-associated virus type 2 (AAV) during **recombinant** vector production. The extent of contamination with wt AAV has been documented to range between 0.01 and 10%. However, the precise mechanism of generation of the contaminating wt AAV remains unclear. To characterize the wt AAV genomes, **recombinant** viral stocks were used to infect human 293 cells in the presence of **adenovirus**. Southern blot analyses of viral replicative DNA intermediates revealed that the contaminating AAV genomes were not authentic wt but rather wt AAV-like sequences derived from **recombination** between (i) AAV inverted terminal repeats (ITRs) in the **recombinant** plasmid and (ii) AAV sequences in the helper plasmid. Replicative AAV DNA fragments, isolated following amplification through four successive rounds of amplification in **adenovirus**-infected 293 cells, were molecularly cloned and subjected to nucleotide sequencing to identify the **recombinant** junctions. Following sequence analyses of 31 different ends of AAV-like genomes derived from two different **recombinant** vector stocks, we observed that all **recombination** events involved 10 nucleotides in the AAV D sequence distal to viral hairpin structures. We have recently

documented that the first 10 nucleotides in the D sequence proximal to the

AAV hairpin structures are essential for successful replication and encapsidation of the viral genome (X.-S. Wang et al., J. Virol. 71:3077-3082, 1997), and it was noteworthy that in each **recombinant** junction sequenced, the same 10 nucleotides were retained. We also observed that **adenovirus** ITRs in the helper plasmid were involved in illegitimate **recombination** with AAV ITRs, deletions of which significantly reduced the extent of wt AAV-like particles. Furthermore, the combined use of **recombinant** AAV plasmids lacking the distal 10 nucleotides in the D sequence and helper

L18 ANSWER 11 OF 11 MEDLINE

ACCESSION NUMBER: 88275039 MEDLINE

DOCUMENT NUMBER: 88275039 PubMed ID: 3292790

TITLE: High-level eucaryotic in vivo expression of biologically active measles virus hemagglutinin by using an adenovirus type 5 helper-free **vector** system.

AUTHOR: Alkhatib G; Briedis D J

CORPORATE SOURCE: Department of Microbiology and Immunology, McGill University, Montreal, Quebec, Canada.

SOURCE: JOURNAL OF VIROLOGY, (1988 Aug) 62 (8) 2718-27.
Journal code: KCV; 0113724. ISSN: 0022-538X.

PUB. COUNTRY: United States

Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 198808

ENTRY DATE: Entered STN: 19900308

Last Updated on STN: 19900308

Entered Medline: 19880819

AB The entire measles virus (MV) hemagglutinin (HA)-coding region was reconstructed from cloned cDNAs and used as part of a hybrid

transcription

unit to replace a region of the adenovirus type 5 genome corresponding to the entire Ela transcription unit and most of the Elb transcription unit. The resulting recombinant virus was stable and able to replicate to high titers in 293 cells (which constitutively express the complementary **Ela-Elb** functions) in the absence of helper virus.

During infection of 293 cells, the hybrid virus expressed MV HA protein which was indistinguishable from that expressed in MV-infected cells in terms of immunoreactivity, gel mobility, glycosylation, subcellular localization, and biologic activity. Infection of 293 cells with the hybrid virus led to high-level synthesis of the MV HA protein (equivalent to 65 to 130% of the level seen in MV-infected cells). At late times

after

high-multiplicity hybrid virus infection of HeLa and Vero cells (which do not express E1 functions), the level of HA protein synthesis was at least 35% of that seen in 293 cells. This MV-adenovirus recombinant will be useful in the study of the biologic properties of the MV HA protein and

in

assessment of the potential usefulness of hybrid adenoviruses as live-virus vaccine **vectors**.